



UNCOMMON COMPLICATIONS OF PANCREATITIS – A CASE SERIES

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ABSTRACT

Pancreatitis is a condition associated with local and systemic complications. Here is a case series comprising of three cases of complicated acute pancreatitis. Two patients had vascular complications in the form of hepatic artery pseudoaneurysm and superior sagittal sinus thrombosis. Another patient developed right sided pleural effusion. All the three complications are extremely rare and the case series is presented along with a brief discussion and literature review.

KEYWORDS : Pancreatitis, pseudoaneurysm, pleural effusion, venous sinus thrombosis

INTRODUCTION:

Acute pancreatitis is inflammatory disorder with local and systemic complications. We report two cases of vascular complications i.e. pseudoaneurysm formation in right hepatic artery and thrombosis in superior sagittal sinus and one case of massive right sided pleural effusion. Vascular complications and significant right sided pleural effusion are very rarely found in cases of pancreatitis.

CASE SERIES:**Case 1. Hepatic artery pseudoaneurysm:**

57 years old gentleman was admitted with central pain abdomen with radiation to back, associated with vomiting. There was no history of diabetes, hypertension or chronic alcohol consumption. Physical examination was unremarkable except anaemia. On the basis of laboratory data [amylase of 236 IU/L (15-100 IU/L) and lipase of 290 IU/L (0-60 IU/L)], he was diagnosed as a case of pancreatitis. The initial abdominal ultrasound revealed bulky pancreas. During the hospital stay patient developed hypotension and acute kidney injury and significant anaemia requiring transfusion. Acute kidney injury resolved with conservative treatment. He underwent upper gastrointestinal endoscopy and it was normal. Follow up ultrasound revealed heterogeneous lesion in caudate lobe of liver. On triple phase contrast enhanced CT of abdomen, there was aneurysmal dilation of right hepatic artery at porta with perilesional oedema (figure - 1) along with features of acute severe pancreatitis with necrosis (CT severity index-7).

The patient was referred to cardiothoracic & vascular surgery department where he underwent endovascular coiling and associated management of the aneurysm.

Case 2. Superior sagittal sinus thrombosis:

43 years old gentleman, chronic alcoholic, was admitted with acute severe pain abdomen radiating to back with vomiting and constipation. Examination reveals tachycardia, tachypnoea, distended abdomen with sluggish bowel movement. Amylase and lipase were 569 U/L and 401 U/L respectively. Conjugated hyperbilirubinaemia was found with mildly elevated liver enzymes. Prothrombin time was normal. Contrast enhanced CT abdomen showed features of mild acute pancreatitis with CT severity index of 2.

During the course of hospital stay he became unconscious which was preceded by severe headache for 4-5 hours. CT brain was done

and suggestive of superior sagittal sinus thrombosis which was confirmed by MR venography (figure:2).

The patient was then treated with low molecular heparin along with other conservative measures for pancreatitis and alcoholic hepatitis. Patient gradually regained consciousness with resolution of other features of pancreatitis.

Case 3. Predominant right sided pleural effusion in asymptomatic pancreatitis:

55 years old gentleman was admitted in ICU of outside hospital with severe shortness of breath with respiratory failure due to right sided massive pleural effusion since about 7 days prior to admission in our hospital. He was on 4 drugs anti-tubercular therapy since then. He had diabetes mellitus & hypertension for which he was on glimepiride, metformin & telmisartan at home. He consumed alcohol regularly.

Over the last 3 to 4 weeks, there was history of pleuritic chest pain predominantly of right side, non-productive cough followed by progressive respiratory distress, and significant weight loss (>6kg over last one month). Examination revealed predominantly right sided pleural effusion without any other systemic abnormality.

There was past h/o lung abscess 8 years back and acute pancreatitis with pseudo pancreatic cyst 1 year back which was resolved on conservative treatment.

Laboratory examination revealed neutrophilic leucocytosis, mild anemia with high ESR. Fasting blood sugar, liver and renal biochemical parameters were within normal range. Pleural fluid cell count was 600, with 80% lymphocyte, 5% mesothelial cells, sugar-110mg%, protein - 4.4 gm. %, LDH - 706 u/l, ADA - 27 I.U.(normal<30 I.U.). No malignant cell was seen. TB PCR was also negative. Chest X ray (figure 3) & contrast enhanced CT thorax were suggestive of bilateral pleural effusion.

As there was no clinical improvement with anti tubercular drugs, we planned for pleural fluid amylase to rule out pancreatic effusion. It was very high (right -1213 u/l; left 1015 u/l). Serum LDH was 571u/l, amylase 413 u/l (normal >120 u/l), lipase 330 u/l (normal >60 u/l). Contrast enhanced CT abdomen was suggestive of pancreatitis without any evidence of necrosis or pseudocyst formation. No evidence of any mass or lymphadenopathy was there in thorax as well as in abdomen. Magnetic Resonance Cholangio Pancreaticography (MRCP) was done to check for any fistulas

connection between pancreatic duct and pleural space but no such connection was seen. Upper GI endoscopy was normal.

So the final diagnosis of pancreatitis with pancreatic pleural effusion was made and treated conservatively. Anti tubercular drugs were stopped. The patient gradually improved and was later discharged.

DISCUSSION:

Disease course of pancreatitis may be affected by local and systemic complication in the form of pseudocyst, pseudo-aneurysm formation, venous thrombosis, pleural effusion and multi organ failure (1). Around 25% cases develop vascular complications like pseudo-aneurysm formation and rupture, haemorrhage within pseudocyst, erosion of arteries, venous thrombosis and formation of varices(2).

We experienced a case of pseudo-aneurysm formation of right hepatic artery which is very rare event in acute pancreatitis. Very few cases had been reported in the past (3, 4). Pseudo-aneurysms due to pancreatitis occur in the splenic artery, 30% in the gastroduodenal artery, 20% in the pancreaticoduodenal artery, 5% in the gastric artery, and 2% in the hepatic artery (5). Mechanism of pseudo-aneurysm formation is thought to occur because of auto digestion by pancreatic enzymes specially elastase liberated by the inflamed pancreas (6). Though gold standard investigation for detecting pseudo-aneurysm is angiography, USG and contrast CT scan are often diagnostic and may be helpful for early diagnosis(7). Otherwise diagnosis usually is delayed and disease course may be complicated with fatal haemorrhage (6). They can be treated with both open surgical techniques & endovascular techniques (6).

Second case as a complication of acute pancreatitis is of superior sagittal sinus thrombosis. Portosplenic and splanchnic venous sinus thrombosis are well described as complications of acute pancreatitis. But extrasplanchnic thrombosis is very rare, particularly superior sagittal sinus thrombosis; which is not described in any literature (8). Systemic inflammatory response associated with pancreatitis is a prothrombotic state and causes endothelial injury and thus participates in the formation of thrombosis (10). Treatment of venous thrombosis in the setting of pancreatitis is doubtful. Splenic vein is the most common site for thrombosis, and chance of spontaneous recanalization is about 30% (9). Anticoagulants are to be started if there is propagating clot. Management of cerebral venous sinus thrombosis in the setting of pancreatitis has yet not been described.

We reported another uncommon complication, right sided pleural effusion in asymptomatic pancreatitis. Incidence of pleural effusion in acute pancreatitis is around 50% as detected by CT scan (11). The effusions were bilateral in 77%, left sided in 16%, and right sided in 8% (11). However pleural effusion in chronic pancreatitis is rare. It is often massive. Mostly the effusion is unilateral and left sided, but approximately 20% are unilateral and right sided. In 15% cases, pleural effusion is bilateral (12, 13). Most patients with chronic pancreatic pleural effusion are men. In more than 90% of male patients, the pancreatic disease is a result of alcoholism (12, 13). Chest symptoms usually dominate the clinical picture of the patient with chronic pancreatic disease with pleural effusion (13).

The diagnosis of pancreatic pleural effusion should be suspected in a patient with pleural effusion who is chronically ill and has prior history of pancreatitis (14). Patients however may not have prior history of pancreatitis (12). The best screening test for pancreatic pleural effusion is pleural fluid amylase which is markedly elevated (usually > 1000 IU/L) whereas the corresponding serum amylase may be normal or mildly elevated (14). Elevated pleural fluid amylase level is seen in pancreatic pleural effusion, malignant diseases and oesophageal rupture. But in malignant effusion the cytology is frequently positive for malignancy and usually amylase level remains < 1000 U/L.

The diagnosis is established by CT abdomen and thorax which

frequently shows both the pseudocyst and the sinus tract (12). In about one third of the patients, fistulous communications could not be demonstrated(15). Endoscopic retrograde cholangiopancreatography (ERCP) also plays an important role in the evaluation and management and also planning for surgical repair (16). Magnetic resonant imaging is complementary to these modalities(16).

The patient of bilateral pleural effusion presented with predominantly chest symptoms with paucity of abdominal symptoms. Diagnosis of pancreatic effusion is made by pleural fluid amylase with virtually excluding all other aetiology of markedly elevated pleural fluid amylase. MRCP was performed to demonstrate ductal rupture but it shown only mild thickening of pancreatic tail.

In conclusion, it is suggested that the pleural fluid amylase content should be measured in any case of recurrent exudative pleural effusion of unknown aetiology.

FIGURE 1: Aneurysm in right hepatic artery with oedema of caudate lobe

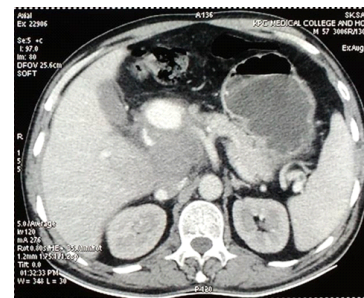


Figure 2: Non occluding recanalised thrombus in the superior sagittal sinus proximal to the confluence of sinuses

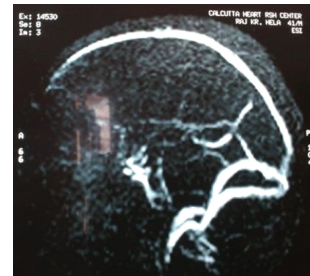
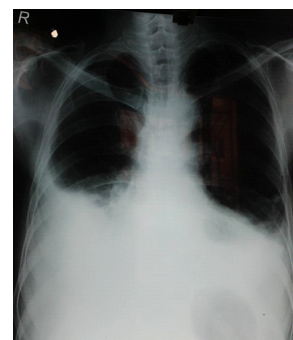


Figure 3: Chest X-ray showing bilateral pleural effusion(Rt>Lt).



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